Introduction

The world wide incidence, morbidity and mortality of allergic asthma are increasing ***(Wills-Karp et al., 1998).*** Asthma is a complex disorder characterized by airway hyperesponsiveness (AHR) and inflammation ***(Jaffar et al., 1999).***

T cell activation and alteration of cytokine levels are involved in the pathogenesis of bronchial asthma ***(Lee et al., 2001).*** Cytokines produced by T–helper type (Th2) lymphocytes have been implicated in airway inflammation and AHR ***(venkayya et al., 2002).*** Among them, the importance of interleukin-4 (IL-4) and IL-13 has emerged, based on the analysis of cytokines expression profiles in lesions, model mice, genetic factors and responses to newly developed reagents ***(Izuhara et al., 2001).*** There is a strong support for the idea that Th2 cytokines can produce AHR indirectly by promoting the recruitment of inflammatory cells ***(venkayya et al., 2002).***

IL-4 is an important cytokine in the allergic inflammation associated with atopic asthma. IL-13 shares many of the biological effects of IL–4 ***(Kotsimbos et al., 1996).*** IL-13 is a pleiotropic cytokine, produced in large quantities by activated $CD4^{+}$Th2 lymphocytes ***(Zhu et al.,1999).*** In animal models of asthma, IL-13 induces goblet cell metaplasia, eosinophil infiltration of the bronchial mucosa, and bronchial hyperreactivity but the basis of its effects on airway epithelium remains unknown ***(Laoukilli et al., 2001).*** Also, the profile of circulating T-Lymphocyte subsets and related cytokines during acute asthmatic attacks is still unclear ***(Lee et al., 2001).***

The aim of this work is to :-

Assess the role of serum level of IL-13 in the pathogenesis of bronchial asthma, in a trial to pave the way for the invention of an-antidote which can either stop or ameliorate the effects of the disease and its sequel.